Bennet Omalu
PATHOLOGY

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Autopsy and Anatomic Pathology
Clinical Pathology and Toxicology
Forensic Pathology

Neuropathology
Epidemiology
Medico-Legal Consultations

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Dear Mr. Faraj,

Re: MUHAMMAD ABDUL MUHAYMIN, DECEASED
MEDICO-LEGAL REPORT

Summary of Education, Training and Experience
I completed medical school in 1990 at the University of Nigeria, Enugu, Nigeria. Upon graduating from medical school, I completed a one-year clinical housemanship at the University of Nigeria Teaching Hospital in the fields of pediatrics, internal medicine, general surgery, obstetrics, and gynecology. After housemanship, I worked as an emergency room physician at a university hospital in Nigeria for approximately three years. I sat for and passed my United States Medical Licensing Examinations [USMLE] while I worked as an emergency room physician. I came to the United States in 1994 through a World Health Organization scholarship to become a visiting research scholar for eight months at the Department of Epidemiology, Graduate School of Public Health, University of Washington, Seattle, Washington.

In 1995, I proceeded to the College of Physicians and Surgeons of Columbia University, New York, New York, at Harlem Hospital Center, to complete residency training in Anatomic Pathology and Clinical Pathology. In 1999 I proceeded to the University of Pittsburgh, Pittsburgh, Pennsylvania to complete residency training in forensic pathology and neuropathology. I hold four board-certifications in Anatomic Pathology, Clinical Pathology, Forensic Pathology and Neuropathology. I also hold a Masters in Public Health [MPH] degree in Epidemiology from the Graduate School of Public Health, University of Pittsburgh, Pittsburgh, Pennsylvania. I also hold a Masters in Business Administration [MBA] degree from the Tepper School of Business, Carnegie Mellon University, Pittsburgh, Pennsylvania, one of the leading business schools in the world. I am a Certified Physician Executive and an honorary fellow of the American Association of Physician Leadership [AAPL]. I also hold a fifth board-certification in medical management from the AAPL. I am licensed to practice medicine and surgery in four states in the United States namely Hawaii, California, Indiana, and Pennsylvania.

I am currently the President and Medical Director of Bennet Omalu Pathology [BOP], a California medico-legal consulting firm, and a Clinical Professor at the Department of Medical Pathology and Laboratory Medicine, University of California, Davis. In my capacity as the Medical Director of BOP, I am a consulting forensic pathologist and neuropathologist to many hospitals in central California and to several counties in northern California. There are less than

a few dozen practicing forensic pathologists-neuropathologists in the United States who are board-certified in both forensic pathology and neuropathology.

For over nineteen years, I have been involved in over nine thousand death and injury investigations in my career as a forensic pathologist and neuropathologist, which began in 1999. I have personally conducted and performed over eight thousand autopsies and death investigations and examined over nine thousand brain tissue specimens. I also perform trauma pattern analysis in both living patients and deceased patients to determine causes and mechanisms of sustenance of injuries and death. I am also involved in the evaluation of living victims of all types of injuries and trauma, including but not limited to victims of assault, traumatic falls, industrial and accidental injuries, medical complications and misadventures, rape, child abuse and sports-related injuries. I have been consulted and retained as an expert witness in one to two thousand cases involving all types of medico-legal cases across all jurisdictions in the United States including federal, state, county and municipal courts and arbitration panels; in both civil and criminal cases, for the plaintiff, defense, district attorneys and public defenders. I have been involved as an expert witness in complex class action and industrial lawsuits involving thousands of individuals and major corporations.

My areas of interest and focus include brain patho-physiology, brain injuries and brain trauma, in both living and deceased patients. I identified Chronic Traumatic Encephalopathy [CTE] in a retired football player when I performed an autopsy and examined the brain of Mike Webster in 2002. Subsequently, I identified CTE in other high-impact, high-contact sports athletes and in military veterans suffering from Post-Traumatic Stress Disorder [PTSD]. Since 2002 CTE has received international attention from the sports industry, sports medicine, and neuroscience. My work has been featured extensively in all media platforms across the world. My work and life were featured in a major Hollywood film, "Concussion" released in December 2015 by Sony Motion Pictures, in which the renowned actor, Will Smith, played me as Dr. Omalu. Several New York Times best-selling books have also been published on my life and work including "The League of Denial" and "Concussion". I have published several books including my memoir, "Truth Doesn't Have a Side", which was published in August 2017. My latest book, "Brain Damage in Contact Sports" was published in February 2018. I have published extensively in the medical and scientific literature authoring many scientific papers and book chapters.

I have received three honorary PhD degrees from two universities in the United States, and from the Royal College of Surgeons of Ireland in recognition of my work and expertise. I have also received numerous awards from across the world in recognition for my work and expertise in both living and deceased patients. I have received the "Distinguished Service Award" from the American Medical Association [AMA], which is the most prestigious award of the AMA. I have been honored by the United States Congress and I have appeared on multiple occasions before committees of the United States Congress and committees of State Legislatures across the Unites States advising them on matters relating to trauma. In 2019 I was appointed to the Traumatic Brain Injury Board of the State of California to advise the state on matters relating to traumatic brain injuries.

Since 1999 I have testified as an expert witness in matters relating to all types of injuries and deaths in over 500 court proceedings across the United States. I have attached a copy of my curriculum vitae, which enumerates my body of work and experience in greater detail. I have also attached my fee schedule. The cases I have testified in, beginning in 2009, are enumerated at the end of my curriculum vitae.

Pursuant upon your request, I have reviewed the following materials in the case of Muhammad Abdul Muhaymin, Deceased:

1. Autopsy and Medical Examiner Reports
2. Body Camera Videos
3. Deposition Transcripts and Recorded Interviews
4. Complaints and Other Miscellaneous Legal Documents
5. Incident Report from the Phoenix Police Department
6. Trip sheet from the Phoenix Fire Department

Summary of Prevailing Forensic Scenario

At the time of his death on January 4, 2017, Muhammad Abdul Muhaymin [Muhammad] was a 43-year-old African-American male who was born on July 14, 1973. He suffered from Post-Traumatic Stress Disorder, Claustrophobia and Schizophrenia. He always carried a service dog with him to alleviate his mental disabilities and symptoms. The dog was named "Chiquita".

Anthony Tarago, the manager of the Maryvale Community Center, called for the police on January 4, 2017 at approximately 09:28 a.m. for an assault and for the police to escort Muhammad from the center and instruct him never to return. Allegedly Muhammad was at the center with his service dog that was off-leash. Muhammad wanted to use the public bathrooms to defecate. Mr. Tarago stood in Muhammad's way and would not permit him to use the bathroom because his dog was not restrained. Allegedly an argument ensued, and Muhammad and Mr. Tarago bumped into each other without any assault.

After the police arrived at the center at approximately 09:32 a.m. Muhammad was allowed to use the bathroom. Officers discovered that there was a misdemeanor warrant for the arrest of Muhammad out of the city of Mesa. The police officers planned to arrest Muhammad after he used the bathroom. Officers walked behind Muhammad as he exited the center carrying his dog in his right arm. An officer grabbed Muhammad's left wrist from the back in an attempt to place him under arrest. A struggle ensued. Muhammad was taken to the ground by the officers at approximately 9:45 a.m. Muhammad clasped his hands together locking them. He did not punch or hit any officer. At least three officers were on top of Muhammad or holding him down, as they pressed him to the ground while attempting to release his hands and handcuff them. The officers placed the weight of their bodies on Muhammad's head, trunk, arms, and legs. Muhammad was heard yelling "Okay!". Two handcuffs were placed, one on each wrist and were linked together behind Muhammad's back.

The officers stood Muhammad up after he was detained at approximately 09:47 a.m. and walked him to a police vehicle at the parking lot. At the police vehicle, Muhammad's handcuffed upper extremities were lifted from behind above his head to the front, while he screamed. Then Muhammad was again taken to the ground by officers at approximately 09:56 a.m. He was placed face down on the sidewalk while officers were on top of him pressing him to the ground, including an officer who used his entire body and laid perpendicularly across Muhammad's right and left calves to keep his legs from moving. Another office knelt on Muhammad's trunk applying his body weight on Muhammad. Muhammad was heard yelling "I can't breathe" several times. A call was made requesting additional officers in order to hold down Muhammad. A RIP [leg] restraint was applied to his feet and around his ankles and then attached to the handcuffs. The officers placed the weight of their bodies on Muhammad's head, trunk, arms, and legs. After Muhammad was re-handcuffed, his body became limp and he began to vomit,

and was moved away from the vomit. He stopped breathing and no pulse was detected at approximately 10:04 a.m. The RIP restraints were removed from his handcuffs and CPR was initiated. Muhammad never struck or kicked any officer. No conducted electrical devices were utilized by any officer and no strikes were deployed by any officer. There were at least six officers who were on top of Muhammad or pressed Muhamad prone on the sidewalk.

Muhammad was emergently transported to the Maryvale Hospital and arrived at approximately 10:23 a.m. Muhammad was unresponsive, had no pulse and was foaming in his mouth. Muhammad was pronounced dead at 10:39 a.m.

Autopsy

A full autopsy [17-00095] was performed on the body of Muhammad Abdul Muhaymin on January 5, 2017 by Dr. Amanda E. Maskovyak at the Maricopa County Office of the Medical Examiner beginning at approximately 10:12 a.m.

Dr. Maskovyak stated the cause of death to be the following: “Cardiac arrest in the setting of coronary artery disease, psychiatric disease, acute methamphetamine intoxication, and physical exertion during law enforcement subdual”. The manner of death was stated to be a homicide. Under the heading “How Injury Occurred”, Dr. Maskovyak stated the following: “Cardiac arrest in the setting of coronary artery disease, psychiatric disease, acute methamphetamine intoxication, and physical exertion during law enforcement subdual”.

The autopsy findings were stated as follows:

I. Cardiac arrest in the setting of coronary artery disease, psychiatric disease, acute methamphetamine intoxication, and physical exertion during law enforcement subdual.
   - A. Coronary artery disease.
     1. 40 to 50 percent atheromatous stenosis of left anterior descending artery.
     2. lntramyocardial segment, left anterior descending artery (2 cm in length x 0.1 cm depth).
   - B. Reported history of schizophrenia and bipolar disorder.
   - C. Acute methamphetamine intoxication.
     1. 0.81 mg/L methamphetamine in iliac blood.
     2. 0.24 mg/L amphetamine in iliac blood.
   - D. History of subdual by law enforcement.
     1. Physical exertion/struggle with officers during law enforcement subdual.
     2. Placement in prone position with hand, knee, foot, and/or body weight force applied by multiple officers to decedent's head, arms, wrists, shoulders, torso, and/or legs.
     3. Physical restraints applied to decedent while in prone position.
     4. Sudden unresponsiveness while in prone position.
     5. Blunt force injuries of head, torso, and extremities.
        - i. Multifocal cutaneous abrasions of face.
        - ii. Bilateral temporalis muscle contusions (right = 2 cm, left = 0.5 cm).
        - iii. No internal head or neck injuries are identified.
        - iv. Intramuscular contusion of left upper back.
        - v. Subcutaneous hemorrhage of left mid back.

vi. Mildly hemorrhagic fractures of anterolateral right and left ribs 2 through 6 and parasternal left ribs 3 through 6 and parasternal right ribs 2 through 6 (possibly representing perimortem artifact due to cardiopulmonary resuscitation with chest compressions).
vii. Multifocal contusions and abrasions of bilateral elbows and wrists.
viii. Subcutaneous and intramuscular hemorrhage of left posterior upper arm and left elbow.
ix. Bilateral anterior knee abrasions.

II. Reported history of asthma.
A No significant histologic evidence of acute asthma exacerbation on histologic examination of lung tissue.

At autopsy Muhammad weighed 164 pounds and measured 67 inches. Vomitus was noted in his nostrils and mouth. The body revealed the following evidence of trauma:

I. Blunt Force Trauma of the Head and Neck:
1. There was a 3 x 1.5 cm orange-pink abrasion of the right lateral frontal scalp.
2. There was a 0.8 cm thin partial thickness linear laceration of the right forehead, just above the right lateral supraorbital ridge.
3. There were 3 x 1.5 cm and 1.2 x 0.8 cm pink contusion and pink-white abrasion of the right lateral supraorbital ridge, respectively.
4. There were two 0.3 cm shallow abrasions of the right upper middle and lateral eyelid.
5. There were 4 x 3 cm clustered and overlapping pink-orange linear abrasions of the right cheek.
6. There was a 0.5 cm laceration of the medial left nare.
7. There was a 2 x 1.5 cm irregular brown abrasion of the left frontal scalp at the hairline.
8. There was a 1.5 cm linear abrasion of the right frontal scalp.
9. There was a 2 x 0.5 cm red-brown abrasion of the left temple.
10. There was a 7 x 1.5 cm cluster of overlapping pink-tan linear abrasions of the left cheek.
11. There was a 2 cm intramuscular contusion of the right temporalis muscle.
12. There was a 0.5 cm intramuscular contusion of the left temporalis muscle.

II. Blunt Force Trauma of the Trunk:
1. There were mildly hemorrhagic fractures of the anterolateral right and left 2nd to 6th ribs and the parasternal left 3rd to 6th ribs, and right 2nd to 6th ribs.
2. There was a 2 cm intramuscular contusion of the left paraspinal upper back.
3. There was a 1.0 cm contusion of the left paraspinal mid back.

III. Blunt Force Trauma of the Extremities:
1. There was a 2.5 x 2 cm red-brown abrasion of the superior left shoulder.
2. There was a 2 x 1.5 cm irregular red-pink abrasion of the left elbow.
3. There was a 2 x 2 cm discontinuous cluster of pink-brown abrasions of the left medial elbow.
4. There was a 2 x 1.5 cm red-brown-pink abrasion of the left posterolateral proximal forearm.

5. There was a 1.0 x 0.9 cm pink-white abrasion of the left lateral wrist.
6. There was a 4.5 x 1.5 cm cluster of irregular pink contusions and abrasions of the left medial wrist.
7. There was a 3.5 x 1.5 cm faint pink contusion of the left volar wrist.
8. There was an 8.0 cm area of ill-defined subcutaneous and intramuscular hemorrhage in the left posterior upper arm.
9. There was a 1.2 x 1 cm red-brown abrasion of the superior right shoulder.
10. There was a 4 x 3 cm cluster of red-brown punctate abrasions of the right medial elbow.
11. There was a 3 x 2.5 cm discontinuous tan-pink abrasion of the proximal right forearm.
12. There was a 2 x 0.5 cm cluster of red-brown abrasions of the right lateral elbow.
13. There were three linear abrasions of the right distal and lateral upper arm measuring 0.3 cm, 0.5 cm, and 1.5 cm.
14. There were 9 x 4 cm discontinuous clusters of pink contusions of the right dorsolateral proximal to mid forearm situated in a vaguely Y-shaped distribution.
15. There was a 4.5 x 4 cm area of faint pink contusions of the right lateral wrist.
16. There was a 3.5 x 3 cm irregular area of pink contusions and abrasions of the right medial wrist.
17. There was a cluster of three irregular abrasions of the proximal medial right forearm measuring between 0.2 cm and 0.7 cm.
18. There were patchy subcutaneous and intramuscular hemorrhages in the right elbow.
19. There was a 1.2 x 0.5 cm irregular red-brown abrasion of the left knee.
20. There was a 0.3 cm red-brown abrasion of the medial left knee.
21. There is a cluster of red-brown abrasions of the right medial and inferior knee measuring between 0.2 cm and 0.8 cm.

The skull revealed no fractures. There were no intracranial hemorrhages. The brain weighed 1300 grams. the heart weighed 325 grams. The left anterior descending coronary artery revealed atherosclerosis with focal 40-50% stenosis. There was tunneling of the left anterior descending coronary artery for a distance of 2 cm and a maximum depth of only 0.1 cm. The right and left lungs each weigh 475 grams. The liver weighed 1425 grams.

Toxicologic analysis of iliac blood obtained during the autopsy revealed the presence of Methamphetamine [0.81 mg/L] and Amphetamine [0.24 mg/L].

Histology Tissue Slides

Seven Hematoxylin and Eosin [H/E] stained tissue histology slides were sent to me from the Maricopa County Office of the Medical Examiner. These were the histology slides prepared after the autopsy and reported in the autopsy report. Based on my education and experience, these seven slides were grossly inadequate and insufficient for accurate pathology diagnoses and conclusions. I therefore requested permission to visit the medical examiner's office and examine the archival autopsy stock tissues in order to prepare additional histology tissue slides. Oddly, the archival autopsy stock tissues fixed in formalin were sent to my office in California. I examined them and made a total of 39 new and additional H/E stained tissue histology slides on this case. I examined all the H/E stained tissue slides, a total of 46 histology tissue slides.

The following tissue slides were examined:

Maricopa County Slides[1]: Seven H/E stained slides[2] without any numbers.
The slide labeled "LAD" reveals a coronary artery with surrounding epicardial adipose tissues and myocardium. The artery shows no significant atherosclerosis with none to minimal eccentric fibrous hyperplasia of the tunica intima. The myocardium shows scattered myofibrillary hypertrophy with box-car nucleomegaly without any inflammation. There is multifocal myofiber hypereosinophilia with myofiber waviness. Another slide shows three small sections of lung tissue, which show diffuse marked congestion without any bronchial or bronchiolar, or alveolar inflammation. There are large amounts of intraluminal aspirated gastric contents and foreign matter in bronchioles. Another slide shows a section of the mesial temporal lobe and cornu ammonis showing diffuse marked congestion of the arachnoidal and penetrating parenchymal blood vessels with perivascular microextravasates in the Virchow Robin spaces. There are many pyknotic and amphophilic to eosinophilic pyramidal neurons in the stratum pyramidalis especially in the Sommer's sector and accentuated in the Sommer's sector. The fascia dentata shows many pyknotic, amphophilic to eosinophilic granule neurons. The inferior temporal cortex also shows many scattered pyknotic, amphophilic to eosinophilic neurons. Another slide shows three small sections of myocardium. The myocardium shows severe vascular congestion with multifocal microextravasates. There are few scattered hypertrophic myofibers with box-car nucleomegaly. There are multifocal and sparse wavy fibers and hypereosinophilic myofibers without any inflammation. Another histology slide shows two small sections of the myocardium and the myocardium shows similar findings described above. Another histology slide shows a small section of the liver and a small section of the kidney. Both sections show diffuse marked parenchymal congestion. The liver section shows no inflammation of the portal triads or lobules and no hepatocellular steatosis or necrosis. The kidney sections show no glomerulosclerosis, interstitial nephritis, or tubular necrosis. Another histology tissue section shows two small sections of lung tissue and both sections reveal diffuse marked congestion without any bronchial or bronchiolar, or alveolar inflammation. There are large amounts of intraluminal aspirated gastric contents and foreign matter in bronchi and bronchioles.

Bennet Omalu Pathology Slides: Thirty-Nine H/E Stained Histology Slides
Slide #1: Two sections of the liver
The liver sections show diffuse parenchymal congestion with no hepatocellular steatosis or necrosis. There is no chronic inflammation of the hepatic portal triads or lobules.
Slide #2: Two sections of the liver
There is focal and sparse lobular hepatocellular infiltration by lymphocytes, Diffuse parenchymal congestion is noted, accentuated in lobular zones 3 in a centrilobular fashion.
Slide #3: Two sections of kidney
There is acute parenchymal congestion with focal micronephrolithiasis in few collecting tubules in the medulla. There is no glomerulosclerosis, interstitial nephritis, or tubular necrosis.
Slide #4: Two sections of kidney
These sections show findings that are similar to what has been described above.
Slide #5: Two sections of lung

[1] The slides were not labeled with any numbers. The inscriptions on all the slides are the following: "17-0095 SUBPOENA AEM Maricopa County Forensic Science Center". Only one slide is also labeled LAD.
[2] The staining of the tissue sections is very faint and faded and are suboptimal for diagnosis.

There is diffuse marked pulmonary parenchymal congestion with focal and sparse mucosa-associated lymphoid tissue without any significant inflammation. There are large amounts of intraluminal aspirated gastric contents in the bronchi and bronchioles.
Slide #6: Two sections of lung
There is diffuse marked pulmonary parenchymal congestion with focal and sparse mucosa-associated lymphoid tissue without any significant inflammation. There are large amounts of intraluminal aspirated gastric contents in the bronchi and bronchioles. Multifocal to patchy to diffuse intra-alveolar eosinophilic edema fluid is noted. Multifocal and sparse intra-alveolar pigment-laden histiocytes are noted.
Slide #7: Two sections of lung
These sections reveal findings similar to what have been described above.
Slide #8: One section of lung; one section of right ventricle
The section of the lung reveals changes, which have been described above. The right ventricle reveals scattered hypertrophied myofibers with box-car nucleomegaly. There is multifocal myofiber waviness and hypereosinophilia [myocytolysis] without any inflammation. There is diffuse congestion of the myocardium.
Slide #9: Two sections of lung
These sections of lung tissue reveal findings, which have been described above.
Slide #10: Two sections of left ventricle
These sections of the left ventricle reveal scattered myofiber hypertrophy with box-car nucleomegaly. There is multifocal myocytolysis with myofiber waviness and hypereosinophilia, with focal contraction band necrosis, without any inflammation.
Slide #11: Two sections of the left ventricle
These sections reveal findings that are similar to what have been described above. There is no inflammation of the endocardium or epicardium.
Slide #12: One section of left ventricle; one section of right ventricle and one section of aorta
The sections of the right and left ventricles show findings which have been described above. Coronary vessels in the epicardium reveal no atherosclerosis. There is no inflammation of the epicardium or endocardium. The aorta shows marked adventitial congestion without any intimal or medial inflammation or necrosis or sclerosis.
Slide #13: One section of the spleen; one section of an adrenal gland
The spleen and adrenal gland show diffuse parenchymal congestion. There is no acute splenitis or adrenalitis. There are no secondary follicles in the splenic white pulp. The adrenal gland shows no hyperplasia or adenoma. There are no adrenocortical or medullary hemorrhages.
Slide #14: One section of pancreas; one section of esophagus and stomach
The pancreas shows early autolysis without any acute or chronic pancreatitis, interstitial fibrosis, or lobular atrophy. The esophagus and stomach show no esophagitis or gastritis.
Slide #15: One section of tongue; one section of cerebellar cortex
The tongue reveals no inflammation and no diffuse myofiber atrophy. The cerebellum reveals diffuse congestion of the arachnoidal and penetrating parenchymal blood vessels with multifocal perivascular microextravasates. The molecular layer shows no necrosis or infarction. The Purkinje neuron layer shows many pyknotic, amphophilic to eosinophilic Purkinje neurons without significant neuronal dropout. The internal granule cell layer shows no autolysis. The cerebellar white matter shows no necrosis, infarct, or demyelination.
Slide #16: One section of interventricular septum; one section of cerebellum
The interventricular septum shows focal myocytolysis with many subendocardial myofibers showing marked hypereosinophilia, myocytolysis and contraction band necrosis without any inflammation. The valvular leaflet attached to the basal interventricular septum shows no inflammation. The cerebellum shows findings, which have been described above in addition to

many scattered pyknotic, amphophilic to eosinophilic neurons in the subcortical nucleus. There is no marked neuronal dropout in the subcortical nucleus. The cerebellar white matter shows marked vascular congestion without any focal necrosis, infarct, or demyelination.
Slide #17: One section of pons; one section of spinal cord:
The section of the spinal cord reveals no inflammation of the meninges or neuropil. The spinal funiculi reveal no degeneration, necrosis, or demyelination. The pons is described below.
Slide #18: One section of pons
The pons is described below.
Slide #19: One section of pons
The pons reveals congestion of the arachnoidal and penetrating parenchymal blood vessels. The basis pontis and pontine nuclei reveal no central myelinolysis. The pontine nuclei reveal scattered pyknotic and amphophilic neurons. The tegmental nuclei and locus ceruleus reveal scattered pyknotic and amphophilic neurons. There is no dorsolateral hemorrhage or necrosis.
Slide #20: One section of medulla oblongata; one section of medulla oblongata-spinal medulla junction
The medulla oblongata reveals no focal neuropil necrosis or hemorrhage. The inferior olivary nuclei reveal mild neuronal dropout with scattered pyknotic and amphophilic neurons. The medullary tegmentum reveals scattered pyknotic and amphophilic neurons without neuropil necrosis. The medullary pyramids show no atrophy, necrosis, or infarct.
Slide #21: One section of medulla oblongata; one section of left ventricle
The medulla oblongata reveals findings, which have been described above. The left ventricle reveals findings, which have been described above.
Slide #22: One section of interventricular septum; one section of prostate gland
The interventricular septum reveals findings, which have been described above. The prostate gland shows sparse and focal acute and chronic prostatitis with neutrophilic and lymphocytic infiltrates. There is no glandular or fibromuscular hyperplasia.
Slide #23: Four sections of spinal cord
There is congestion of the spinal meninges and neuropil without any inflammation. The white matter funiculi, including the anterior and lateral funiculi and the fasciculus cuneatus and gracilis, reveal neuropil edema without focal necrosis, inflammation, or demyelination. The central gray matter shows no hemorrhage and no necrosis. The spinal neurons show no degeneration. The spinal canal is central.
Slide #24: One section of mesial temporal lobe and hippocampus
See description below.
Section #25: One section of neocortex
See description below.
Slide #26: One section of mesial temporal lobe and hippocampus
The dentate gyrus and cornu ammonis of the hippocampus reveal no focal dysplasia or sclerosis. The stratum pyramidalis of the Sommer's sector (CA1), CA2, CA3 and CA4 regions of the cornu ammonis reveal many scattered pyknotic, amphophilic to eosinophilic pyramidal neurons especially in the Sommer's sector. The fascia dentata reveals scattered pyknotic, amphophilic to eosinophilic granule neurons. The entorhinal cortex and the alveus reveal no focal neuropil necrosis. The choroid plexus is congested. The penetrating vessels of the strata radiatum, lacunosum and moleculare reveal mural mineralization.
Slide #27: One section of neocortex
See description below.
Slide #28: One section of neocortex
Examination of hematoxylin and eosin stained sections of the neocortex reveals a homogenetic cortex with the normal homotypical laminar and columnar organization. There is no cortical

disorganization, diffuse or nodular neuronal heterotopia. There is no significant neuronal drop-out or astrogliosis. There are many scattered pyknotic, amphophilic to eosinophilic neurons. There is diffuse congestion of the arachnoidal and penetrating parenchymal blood vessels with multifocal perivascular microextravasates. There is perineuronal vacuolation, accompanied by expansion of Virchow Robin spaces and neuropil microspongiosis of both the gray and white matter. The arachnoidal mater, pia mater, penetrating vessels and the Virchow Robin's spaces reveal no inflammation. The centrum semiovale reveals neuropil edema and congestion without any focal necrosis, infarct, or hemorrhages. There is no gliding contusional hemorrhage.
Slide #29: Sections of dura mater
See the description below.
Slide #30: Section of dura mater
The dura mater reveals intradural congestion with intradural microextravasates without any inflammation, extradural membranes or pigment-laden histiocytes.
Slide #31: Section of larynx
The larynx reveals sparse mucosa-associated lymphoid infiltrates.
Slide #32: One section of larynx; one section of epicardium with coronary artery
The larynx reveals no inflammation. The coronary artery shows eccentric mild fibrous hyperplasia of the tunica intima with focal intramural cholesterol deposits and vacuolation.
Slide #33: One section of epiglottis; one section of trachea
The epiglottis reveals focal and sparse submucosal lymphocytic infiltrates. the trachea does not reveal any inflammation.
Slide #34: One section of adrenal gland; one section of urinary bladder
The adrenal gland shows diffuse congestion without any necrosis or hemorrhage. There is no adenoma or hyperplasia and no inflammation. The urinary bladder reveals no cystitis.
Slide #35:One section of thyroid gland; one section of heart- SA node
The SA node shows no necrosis, dysplasia, fibrosis, or inflammation. The thyroid gland does not show any thyroiditis or nodular hyperplasia.
Slide #36: One section of lung; one section of AV node
The AV node appears unremarkable. The lung section reveals findings, which have been described above.
Slide #37: One section of heart; one section of gallbladder; two sections of vermiform appendix
The heart reveals findings, which have been described above. The gallbladder reveals diffuse autolysis without any inflammation. The vermiform appendix reveals no inflammation.
Slide #38: One section of bronchus; one section of vermiform appendix and mesoappendix; one section of gallbladder
The gallbladder shows diffuse autolysis without any inflammation. The vermiform appendix shows no inflammation. The mesoappendix shows marked vascular congestion. The bronchus shows marked diffuse, transmural congestion without any inflammation.
Slide #39: Four sections of epicardium and myocardium and coronary vessels
The coronary vessels in these sections do not reveal any inflammation. There is none to minimal fibrous hyperplasia of the tunica intima.

Medico-Legal Questions

1. What were the underlying cause of death, mechanism of death, contributory factor to death and manner of death of Muhammad Abdul Muhaymin [Muhammad]?

Medicine is a life science, which is evidence based. The practice of medicine is guided by established standards and generally accepted principles, which certified physicians must adhere to. The specialties and the categories of physicians who are proficiently trained, specialized, and competent in the accurate determination of the cause, mechanism and manner of death are the forensic pathologists, especially for deaths involving all types of trauma and bodily injury. The death of Muhammad involved serious bodily injury.

The College of American Pathologists [CAP] describes the specialty of forensic pathology as follows: "Forensic pathology is the subspecialty of pathology that directs its efforts to the examination of living or dead persons in order to provide an opinion concerning the cause, mechanism, and manner of disease, injury or death; the identification of persons; the significance of biological and physical evidence; the correlation and/or reconstruction of wounds, wound patterns, and sequences; and conducting comprehensive medico-legal death investigations. Forensic pathology applies techniques of pathology to the needs and protection of public health, public safety, quality assurance, education in medicine, research, jurisprudence, and the administration of justice. Its highest goal is the development of strategies to prevent injury, disease, and death."

The CAP also describes a forensic pathologist as follows: "A forensic pathologist is a pathologist with special training and experience in forensic pathology who is actively engaged in medico-legal autopsies and death investigations. Forensic pathologists shall be board-certified by the American Board of Pathology or American Osteopathic Board of Pathology after appropriate training and passing a rigorous examination, or a non-USA based pathologist with equivalent certification. The practicing forensic pathologist is licensed in one or more states; he/she is skilled in conducting death investigations, interpreting injuries in both fatal and non-fatal cases, performing medico-legal examinations, determining disease/injury causation to an appropriate degree of medical certainty and determining cause and manner of death."

Trauma pattern recognition, interpretation and analyses are the fundamental methodologies the forensic pathologist adopts in the differential diagnoses of causes and mechanisms of injuries and/or death. Trauma pattern recognition, translation and analysis are commonly applied to forensic differential diagnoses, opinions, and conclusions. It is a generally accepted principle and common knowledge in medicine and forensic pathology, that specific traumatic events generate predictable, reproducible, and specific patterns of injuries, outcomes, and death.

The practice of forensic pathology is guided by very well-established and generally accepted principles, which board-certified forensic pathologists must adhere to while they routinely perform differential diagnoses and determine causes, mechanisms, and manners of death. Objective decisions, conclusions and opinions should be made in all types of trauma case analyses strictly based on objective interpretations of patterns of injuries, prevailing forensic scenarios, and trauma pathophysiology. The prevailing global forensic scenarios, the patterns of trauma and the expected outcomes of trauma exhibited by Muhammad are vividly consistent

with fatal and homicidal trauma as prescribed by the well-established and generally accepted patho-physiology of trauma and disease.

The determination of cause and manner of death are guided by and must adhere to very well-established and generally accepted principles and concepts, standards of practice and common knowledge of science and medicine. In order to determine the cause of death of Muhammad accurately and competently, we may have to review these generally accepted principles and concepts, standards of practice and common knowledge. Forensic pathologists cannot determine cause and manner of death at whim outside these principles, concepts, and standards when they perform differential diagnoses to determine causes, mechanisms, and manners of death. Objective decisions, conclusions and opinions should be made in all types of trauma case analysis strictly based on objective interpretations of patterns of injuries and prevailing forensic scenarios, which should be based on these well-established and generally accepted principles and concepts, standards of practice and common knowledge of science and medicine.

There are four components of cause of death, viz: underlying cause of death, contributory factor to death, mechanism of death and manner of death.

What is an underlying cause of death?
The underlying cause of death is defined as the single factor, event, or disease, which instigates or initiates a terminal chain of events that finally culminates in death. It must not be a single disease. An event like compression of the body, a gunshot wound of the head, an assault or a fall can be a cause of death, when it initiates the terminal chain of events. The chain of events, which occurs between the underlying cause of death and death itself, encompasses the mechanisms of death. Mechanisms of death are typically not written on the death certificate.

An illustration is when an individual is shot in the spine causing quadriplegia. Assuming the individual survives for fifteen years after he was shot and develops the known sequelae of quadriplegia like recurrent bronchopneumonia, recurrent aspiration pneumonia, recurrent urinary tract infections, and decubitus ulcers, and finally develops an overwhelming sepsis and dies from sepsis. The underlying cause of death will be the event, gunshot wound of the spine. The terminal chain of events and the mechanisms of death would include the recurrent infections and the sepsis, which finally preceded death. Although the immediate causes of death in this instance are natural diseases, the traumatic gunshot wound of the spine precipitated the natural diseases and would supersede the natural diseases. The cause of death in the death certificate may be completed as “Gunshot Wound of the Spine”.

When unnatural events or diseases, like falls from any height, compression of the body or fractures, co-occur with natural events or diseases, like cancer or heart disease in the cascade of events that precipitate death, the unnatural events or diseases supersede the natural events or diseases and assume the cause and manner of death.

An illustration is a 60-year-old woman who is dying from end-stage cancer and has only several months to live. She is admitted into a hospice care center for comfort care only. She got up from bed one morning to go to the bathroom, slipped, fell on the ground, and impacted her head on the floor. She sustained subdural hemorrhages inside her skull and died several weeks later from complications of cancer and surgery to evacuate the subdural hemorrhage. The cause of death in this instance would be the traumatic brain injury she suffered because it was an unnatural disease or event, although she had suffered advanced cancer for several years and was dying

from terminal cancer, which is a natural disease. The manner of death therefore would be an accident.

For a factor or disease to assume the underlying cause of death, there has to be a contiguous chain of events between the initial occurrence of that factor or disease and the occurrence of death, without any significant breach. The interval between the initiating factor and final demise is immaterial and non-contributory to the determination of an underlying cause of death as far as a contiguous chain of events can be established and competently linked to the initiating factor without any significant breach.

When there is a pre-existing lethal chain of events from any factor or disease, and a novel factor or disease arises, either dependent on, or independent of the pre-existing factor or disease, and successfully disrupts and breaches the contiguity of the chain of events of the pre-existing factor or disease, while initiating a novel lethal chain of events, which culminates in death, the novel factor or disease would assume the underlying cause of death.

An illustration is the instance of a 55-year-old obese man with severe coronary atherosclerotic disease, who has had multiple myocardial infarctions and a triple coronary artery by-pass surgery and has developed and is dying from end-stage congestive cardiac failure from ischemic cardiomyopathy. If this same man falls backwards at home while opening a chest of drawers, which falls on top of him, entraps him and compresses his trunk for about 5 minutes before his 26-year-old son finds him and moves the chest of drawers off him. Unfortunately, by this time he was beginning to lose consciousness. The wife calls 911, paramedics arrive and emergently take him to the hospital where he is successfully resuscitated but had suffered asphyxial brain injury. He is admitted into the intensive care unit where he dies two days later from complications of compression of the trunk and asphyxial brain injury. Although he was suffering and dying from severe and advanced heart disease, the compression of his trunk, which he suffered was a novel and independent factor which instigated a novel chain of events, which successfully interrupted the previously existing chain of events, which culminated in his death. Compression of the trunk would therefore assume the underlying cause of death and determine the manner of death, which in this instance would be an accident. The asphyxial injury of the brain is an unnatural disease and would supersede the natural diseases and assume the cause and manner of death.

For every disease, there are extenuating and aggravating factors, which can either decrease or increase the risk of suffering from or dying from a disease. A contemporaneous or co-morbid disease or factor that increases the risk of a second disease or factor does not denote causation, rather it denotes co-morbidity. Disease or event “A” that is co-morbid with disease or event “B” does not mean disease “A” causes disease “B” and vice versa.

<u>What is a contributory factor to death?</u>
A heading in the death certificate states the following: “Other Conditions Contributing to Death”. A contributory factor to death is defined as any factor, disease or event, which occurs contemporaneously with the underlying cause of death, possesses an independent capacity to cause death, however the lethality of this capacity is inferior to the lethality of that of the underlying cause of death. The contributory factor may accentuate or accelerate the lethality of the underlying cause of death.

An illustration is the instance of a man who suffers from end-stage metastatic lung cancer and sustains a fracture of his humerus when he fell in his living room. He is taken to the hospital and he undergoes open reduction and internal fixation with intramedullary rods. He unfortunately suffers a post-traumatic fat embolism following his surgery and dies from acute respiratory failure six days after he sustained his fracture. The underlying cause of death would be acute respiratory failure due to traumatic fat embolism due to fracture of the humerus. The contributory factor to death will be the metastatic lung cancer. The manner of death would be an accident. The lethal capacity of the traumatic fat emboli caused by his fractured humerus is far more superior to the lethal capacity of his lung cancer. This is why the fractured bone killed him within six days while he had survived lung cancer for three years. Traumatic fat embolism from a fractured long bone and metastatic lung cancer independently possess potent lethal capacities, however the lethal capacity of traumatic fat embolism is superior to that of lung cancer; and traumatic fat embolism is an unnatural disease, while lung cancer is a natural disease. Traumatic fat embolism caused by a fracture would therefore become the underlying cause of death and assume the manner of death, which will be an accident. The lung cancer will become the contributory factor to death.

A contributory factor to death may become an underlying cause of death, if and when it instigates a chain of events, which successfully interrupts the pre-existing chain of events of the underlying cause, which has been discussed above. In this instance, the underlying cause of death would become the contributory factor.

What is a manner of death?

The manner of death is a medico-legal terminology, which categorizes the circumstances, which surround death sometimes referred to as “the prevailing terminal forensic scenario”. There are two broad categories of manners of deaths:

1. Natural
2. Un-natural

Natural deaths are deaths caused by known natural diseases as have been published in the International Classification of Diseases. Un-natural deaths are classified into four manners of death:

1. Homicide
2. Suicide
3. Accident
4. Undetermined

For this report, only the homicide manner of death will be defined. A death is classified as a medical homicide when a person intentionally, knowingly, recklessly, or negligently causes the death of another human being. A medical homicide may be deemed as a death that occurs, directly or indirectly, as a result of another person’s actions.

In the determination of manner of death, whenever an un-natural factor plays a role in the causation and mechanism of death, no matter how infinitesimal, the unnatural factor supersedes the natural factors and assumes the manner of death.

The case of Muhammad

On January 4, 2017 Muhammad was a 43-year-old male who was alive and well and was not in any imminent death or was dying from any disease before he encountered the police. He suffered from the psychological ailments of Post-Traumatic Stress Disorder, Claustrophobia and Schizophrenia. His autopsy confirmed that he suffered minimal coronary artery disease, which is prevalent in the United States population and was not a disease that was going to kill him. He was not dying and was not expected to die. Although he had a history of what was believed to be bronchial asthma, autopsy confirmed that he was not suffering from bronchial asthma.

Muhammad was a member of the community and was at a community center to use a public bathroom to answer to a normal physiological call of nature, to pass stool. He did not pose any reasonable threat or danger to himself or society, was not wanted for a felony, and was not a felon who posed a danger to society, and had not committed a felony that was punishable by the death penalty, life imprisonment or a long-term imprisonment.

Muhammad did not die from Methamphetamine toxicity

Toxicologic analysis of his central blood obtained during his autopsy revealed the presence of Methamphetamine [0.81 mg/L] and Amphetamine [0.24 mg/L]. This means that Muhammad had consumed Methamphetamine within hours of his death. Every human being who consumes Methamphetamine, even on habitual basis, does not die from Methamphetamine overdose. Majority of human beings who consume Methamphetamine as a recreational drug do not die from Methamphetamine. For example, statistical estimates indicate that about 13 million people, 12 years and older, in the United States have reported using Methamphetamine. Only about 100 thousand people [0.77%] visit the emergency department for Methamphetamine toxicity annually, and only about 3 thousand people [0.02%] die from Methamphetamine toxicity annually[3]. If not for his encounter with the police on January 4, 2017, more likely than not, Muhammad would not have died, and was not expected to die on January 4, 2017.

Methamphetamine was not the underlying cause of death of Muhammad. The presence of a drug in the blood of a deceased person does not equate to causation of death. The toxic effects of a drug cannot be only or fully deciphered solely based on the level of the drug in the blood. This is outside the generally accepted guidelines and standards of practice of clinical pathology and interpretative toxicologic analysis for the determination of cause of death[4]. The toxic effects of a drug are multifactorial and are determined by many independent and sometimes mutually exclusive factors. However, the level of Amphetamine in Muhammad's blood was stated to be 0.24 mg/L which was about the same level with the expected toxic levels of >0.20 mg/L[5,6]. The level of Methamphetamine in the blood of Muhammad was 0.81 mg/L, which was higher than the expected toxic level of Methamphetamine which is 0.15 mg/L[7,8]. This is why a determination

[3] https://drugabuse.com/library/methamphetamine-history-and-statistics/#how-dangerous-is-methamphetamine-
[4] Ferner RE. Post-mortem clinical pharmacology. British Journal of Clinical Pharmacology, 2008;66(4):430-443.
[5] Schulz M et al. Therapeutic and toxic blood concentrations of nearly 1000 drugs and other xenobiotics. Critical Care, 2012;16:R136.
[6] Regenthal R et al. Drug levels: therapeutic and toxic serum/plasma concentrations of common drugs. Journal of Clinical Monitoring and Computing, 1999, 15:529-544.
[7] Schulz M et al. Therapeutic and toxic blood concentrations of nearly 1000 drugs and other xenobiotics. Critical Care, 2012;16:R136.
[8] Regenthal R et al. Drug levels: therapeutic and toxic serum/plasma concentrations of common drugs. Journal of Clinical Monitoring and Computing, 1999, 15:529-544.

of the cause of death of any individual should not be based on only the level of a drug in the blood.

Chronic use of Methamphetamine results in psychologic dependence and tolerance, therefore, the effects of Methamphetamine may be modulated by the drug use history of each individual. Chronic use and exposure result in chronic drug tolerance and resistance. Muhammad was a chronic drug user, and people who use Methamphetamine chronically develop drug tolerance and habituation and as time passes would use and need higher levels of drugs to achieve the same effects. Therefore, such high levels of Methamphetamine and Amphetamine as documented by the autopsy of Muhammad are expected because he was a chronic user of Methamphetamine.

Moreover, following death, high tissue levels of drugs migrate/diffuse into the blood, especially in chronic consumers of the drugs, and create artifactually and markedly elevated levels of drugs in the blood when measured at autopsy. Methamphetamine exhibits a post-mortem tissue redistribution of 0.9 – 2.4 heart/femoral ratios. Amphetamine exhibits a post-mortem tissue redistribution of 1.2 – 5.6 central/peripheral ratios[9]. This means that the post-mortem levels of Methamphetamine and Amphetamine reported in the autopsy blood sample of Muhammad were actually much higher than the pre-mortem levels prior to his death. These ratios may be higher in chronic users who have accumulated the drug in their tissues over time.

Therefore, post-mortem levels of 0.81 mg/L of Methamphetamine and 0.24 mg/L of Amphetamine may not be of such a significant forensic consequence as it would have been for an individual who has never used Methamphetamine or who has not attained the level of drug tolerance Muhammad had attained as a chronic recreational user. This is exemplified by some cases reported on page 1320 of the textbook: "Disposition of Toxic Drugs and Chemicals in Man", eleventh edition, by Randall C. Baselt, PhD. This page states that "Two women found by police asleep in a car had blood Methamphetamine concentrations of 1.7 mg/L and 2.1 mg/L, as well as substantial levels of Diazepam. The same book on the same page gives two additional examples as follows: [1] Methamphetamine blood concentrations of 1.4 – 13 mg/L [average 5.1 mg/L] were found post-mortem in nine drug abusers who died of traumatic injury by violent means; [2] Blood Methamphetamine concentrations of <0.05 – 2.6 mg/L were observed in 27 persons arrested for erratic driving."

The toxic levels and effects of drugs that physicians use and apply to case management and analysis are typically related to therapeutic drug levels and toxicities. However, the National Highway Traffic Safety Administration reports a non-toxic range of 0.01 to 2.5 mg/L[10] in individuals who use Methamphetamine for recreational purposes. This further confirms that the level of Methamphetamine detected in the blood of Muhammad was of no significant forensic consequence, was not expected to kill him and did not kill him. He used Methamphetamine recreationally and chronically.

[9] Baselt RC, Disposition of Toxic Drugs and Chemicals in Man, 11th edition, Biomedical Publications, Seal Beach, California, 2017.
[10] National Highway Traffic Safety Administration. Drugs and Human Performance Fact Sheets. U.S. Department of Transportation- Methamphetamine [and Amphetamine] www.nhtsa.dot.gov

Muhammad did not die from coronary artery disease

In her autopsy report, Dr. Maskovyak concluded that Muhammad died as a result of coronary artery disease. This is false and grossly inaccurate and goes against the generally accepted principles of cardiology and cardiac pathology. Coronary artery disease causes death through the pathophysiological mechanism of ischemic myocardial injury and myocardial infarction, which can manifest as cardiac arrhythmia and cardiac arrest. According to the international classification of myocardial infarctions[11], there are five types of myocardial infarctions, viz: Types 1, 2, 3, 4 and 5.

Type 1 myocardial infarction is precipitated by an atherosclerotic plaque disruption, rupture, or erosion. The thrombotic component may lead to distal coronary embolization. Plaque rupture is complicated by intraluminal thrombosis and by hemorrhage into the plaque through the disrupted surface. Muhammad did not suffer a Type 1 myocardial infarction.

Type 3 myocardial infarction occurs when a patient presents with myocardial injury or ischemia including presumed new ischemic EKG changes or ventricular fibrillation and dies suddenly and unexpectedly before it is possible to obtain blood or cardiac biomarker determination. Or the patient dies after the onset of symptoms before an elevation of biomarker values has occurred. Autopsy does not show any ruptured or disrupted plaque and no intraluminal thrombosis. Muhammad did not suffer a Type 3 myocardial infarction. Type 3 myocardial infarction allows the separation of fatal myocardial infarction events from the much larger group of sudden death episodes that may be cardiac [non-ischemic] or non-cardiac in origin. Muhammad did not die from a type 3 myocardial infarction.

Type 4 myocardial infarction occurs as a result of cardiac procedural myocardial injury related to percutaneous coronary intervention and revascularization procedures. It may be related to the procedure itself, which may reflect periprocedural issues or complications of a device such as early or late stent thrombosis or in-stent restenosis. Muhammad did not suffer a Type 4 myocardial infarction.

Type 5 myocardial infarction occurs as a result of cardiac procedural myocardial injury related to coronary artery bypass grafting procedures. It may be related to the procedure itself, which may reflect periprocedural issues or complications, graft occlusion or stenosis. Muhammad did not suffer a Type 5 myocardial infarction.

If Muhammad rightfully died as a result of coronary artery disease, he would have suffered a type 2 myocardial infarction. However, it is pertinent to note that Muhammad did not have moderate or severe coronary artery disease. As this scientific article states[12]:
"The pathophysiological mechanism leading to ischemic myocardial injury in the context of a mismatch between oxygen supply and demand has been classified as type 2 myocardial infarction. By definition, acute atherothrombotic plaque disruption is not a feature of type 2 myocardial infarction. In patients with stable known or presumed Coronary Atherosclerotic Disease, an acute stressor such as an acute gastrointestinal bleed with a precipitous drop in

[11] Thygesen K et al. Fourth universal definition of myocardial infarction [2018]. European Heart Journal, 2019;40:237-269.
[12] Thygesen K et al. Fourth universal definition of myocardial infarction [2018]. European Heart Journal, 2019;40:237-269.

hemoglobin, or a sustained tachyarrhythmia with clinical manifestations of myocardial ischemia, may result in myocardial injury and a type 2 myocardial infarction. These effects are due to insufficient blood flow to the ischemic myocardium to meet the increased myocardial oxygen demand of the stressor. Ischemic thresholds may vary substantially in individual patients depending on the magnitude of the stressor, the presence of non-cardiac comorbidities, and the extent of underlying Coronary Atherosclerotic Disease and cardiac structural abnormalities."

"The short- and long-term mortality rates for patients with type 2 myocardial infarction are generally higher than for type 1 myocardial infarction patients in most but not all studies due to an increased prevalence of comorbid conditions. Coronary atherosclerosis is a common finding in type 2 myocardial infarction patients selected for coronary angiography."

"The context and mechanisms of type 2 myocardial infarction should be considered when establishing this diagnosis. The myocardial oxygen supply/demand imbalance attributable to acute myocardial ischemia may be multifactorial, related either to: reduced myocardial perfusion due to fixed coronary atherosclerosis without plaque rupture, coronary artery spasm, coronary microvascular dysfunction [which includes endothelial dysfunction, smooth muscle cell dysfunction, and the dysregulation of sympathetic innervation], coronary embolism, coronary artery dissection with or without intramural hematoma, or other mechanisms that reduce oxygen supply such as severe bradyarrhythmia, respiratory failure with severe hypoxemia, severe anemia, hypotension/shock; or to increased myocardial oxygen demand due to sustained tachyarrhythmia or severe hypertension with or without left ventricular hypertrophy".

It becomes vividly obvious that if coronary artery disease played a role in the death of Muhammad, he would have suffered a Type 2 myocardial infarction instigated by a stressor. The stressor in this instance would be mechanical-positional asphyxiation by sustained compression of the trunk and body and compressed prone-positioning on the sidewalk. This acute stressor instigated a cardiac oxygen demand-supply mismatch, which precipitated ischemic myocardial injury. Coronary artery disease, therefore, cannot be a cause of death in this instance of sudden traumatic death, but a mechanism of death. A mechanism of death cannot be classified as an underlying cause of death and should not be listed on the death certificate.

A type 2 myocardial infarction does not require coronary atherosclerotic disease to be present to occur. The fundamental principle and criterion for a type 2 myocardial infarction is the presence of a stressor, which places the heart at an increased demand for oxygen, and when there is a mismatch between the supply of oxygen and the metabolic rate of the heart cells, myocardial injury and infarction occurs. If coronary artery disease is present, the blood vessels supplying the heart may not dilate sufficiently or have luminae that can open wide enough for larger amounts of blood to pass per unit time and per unit mass of the heart. So, the presence of coronary artery disease is not the underlying cause of a type 2 myocardial infarction but a comorbidity that can contribute to the mechanism of injury of myocardial ischemia and infarction.

Muhammad died as a result of Mechanical-Positional Asphyxiation due to Compression of his Trunk and Body

When Muhammad walked out of the community center on January 4, 2017 he was not in any form of distress and was not dying. A novel factor or event occurred unexpectedly and suddenly in his life at approximately 09:45 a.m. He became unconscious at approximately 10:04 a.m., 19 minutes later. He was pronounced dead at the hospital at 10:39 a.m.

Dr. Maskovyak who performed the autopsy stated the following on page 13 of her autopsy report: "No significant pathologic abnormalities" seen upon microscopic examination of the only section of the brain that she examined, the hippocampus. This is grossly inadequate, insufficient and beneath the standards of practice of forensic pathology and forensic neuropathology. I took many more topographically targeted and anatomically selective sections of the brain and examined them, which I have described above. There is no question whatsoever that Muhammad suffered acute asphyxial brain injury [hypoxic-ischemic brain injury] as manifested by the following findings and diagnoses in the brain:

I. Congestive Brain Swelling, Global, with Diffuse Cerebral Parenchymal Edema, Acute
II. Selective Neuronal Excitotoxic Injury with Selective Topographic Vulnerability

Dr. Maskovyak is not a certified or credentialed neuropathologist and may lack the prerequisite knowledge and expertise to competently identify and accurately diagnose Asphyxial Brain Injury due to Mechanical-Positional Asphyxiation due to Compression of the Trunk and Body, which Muhammad Abdul Muhaymin, a 43-year-old African-American male died from.

The global evidentiary autopsy findings in this case confirm that Muhammad suffered diffuse and global asphyxial brain injury [hypoxic-ischemic brain injury]. The human brain is a post-mitotic organ and can only survive on oxygen and glucose, which are supplied by blood that come from the heart, primarily in the internal carotid arteries and the vertebral arteries. While the brain is only about 2-3% of the body weight, it receives approximately 15% of the cardiac output at a rate of 750-900 ml/min of blood. The normal range of perfusion of the brain is about 50 to 65 ml/100 g/min [80-100 ml/100g/min for the gray matter and 20—25 ml/100g/min for the white matter, at a rate of oxygen consumption of 3.5 ml/100 g/min. The normal brain tissue partial pressure of oxygen is 35 to 40 mmHg. Brain tissue oxygen levels below 30 mmHg may cause brain tissue injury, and at 20 mmHg, the risk of brain damage becomes exponentially elevated. The threshold for brain infarction is 10-12 ml/100g/min of blood supply with neuronal injury and death beginning in 60 to 180 seconds.

Being a post-mitotic organ, the human brain does not have any reasonable capacity to regenerate itself. This means that when the human brain suffers any type of irreversible injury, that injury is permanent and cannot be reversed or cured by the brain or by medical therapy. There are so many types of brain injuries. Asphyxial brain injury [hypoxic-ischemic brain injury] is only one type of brain injury. For the human brain to suffer irreversible hypoxic-ischemic brain injury, there has to be impaired supply of oxygen and blood to the brain for a relatively long period. The established and generally accepted median or mean reference threshold time for irreversible hypoxic-ischemic brain damage to occur is 3 to 5 minutes in cumulative time. This means that irreversible brain damage can occur in less than 3 minutes or in more than 5 minutes, but with a mean or median time of close to 3 to 5 minutes.

The forensic question that arises at this juncture is what caused Muhammad's diffuse, global hypoxic-ischemic brain injury? Any factor that impairs the entire respiratory functioning of the

human being can result in the deprivation of oxygen to the brain. Absolute deprivation [complete absence of oxygen- anoxia] is not necessary for the brain to suffer brain injury. Any factor that impairs the body's ability to inspire oxygen and expire carbon dioxide, impairs the body's ability to bind oxygen to hemoglobin, impairs the body's ability to transmit oxygenated blood to the brain, impairs the brain's ability to absorb oxygen from oxygenated blood, impairs the brain's ability to utilize oxygen, and to transmit carbon dioxide to the blood and transport it out of the brain tissues can result in hypoxic-ischemic injury to the brain. Diminishing levels of oxygen and increasing levels of carbon dioxide in brain tissue impairs the cerebral vascular autoregulation, which results in impaired vascular perfusion of the brain, which causes combined hypoxic-ischemic injury of the brain. This means that the brain does not have to suffer complete lack of oxygen or blood to suffer brain damage. There are multiple metabolic factors that can ameliorate or aggravate the risk of brain damage at varying levels of oxygen, glucose, and blood supply to the brain.

Compression of the trunk and body also causes neurological compression injury to vital nerves and plexuses in the trunk, head and neck, which decreases respiration and systemic blood pressure, which in turn decreases cerebral perfusion pressure, which accentuates the hypoxic-ischemic injury of the brain. When the motor, sympathetic and parasympathetic innervations and systems of the nerves in the trunk and neck, including but not limited to the vagus nerve, phrenic nerve, glossopharyngeal nerve, hypoglossal nerve, cervical sympathetic trunks and cervical parasympathetic ganglia, undergo sustained compression, there is a combined effect of bradycardia, systemic hypotension, cardiac arrest and respiratory arrest. The higher the scale of the compression, the more sustained the compression is and the longer the compression lasts the greater the risk of attaining irreversible neurological injury and the greater the risk of suffering permanent and irreversible end-organ consequences, like we have in the case of Muhammad.

For normal and optimal respiratory activity to occur the thoracic pressure remains at negative atmospheric pressure to support intricate homeostatic undulations of intrathoracic pressure that allow normal and effortless respiratory movements of the diaphragm, chest, and lungs. Compression of the trunk and body as we have in this case undermines this homeostatic balance and increases the risk of respiratory failure, asphyxial injury of the brain and sudden death.

Muhammad was placed prone on the hard floor of a building beginning at approximately 09:45 a.m. on January 4, 2017. At least three officers were on top of him, held him down and pressed him on the hard floor of a building. The officers placed their weights on his head, trunk, and extremities for at least 2 minutes. He yelled "Okay!". The officers placed handcuffs on Muhammad and stood him up from the ground beginning at approximately 09:47 a.m.

Less than ten minutes later, beginning at approximately 09:56 a.m. Muhammad was again placed prone on the hard ground of a sidewalk while officers were on top of him. At least six officers pressed him down on the ground and placed the weights of their bodies on Muhammad's head, trunk, and extremities. Muhammad yelled "I can't breathe" several times. Muhammad's gastro-esophageal sphincter collapsed in response to the high traumatic stressor levels and he vomited and became unresponsive. The officers observed that he was no longer responsive beginning at approximately 10:04 a.m. His body and trunk were mechanically compressed for about eight minutes before he vomited and became unresponsive. Microscopic examination of all the sections of the lungs shows diffuse and marked occlusion of the bronchial and bronchiolar airways by large amounts of aspirated gastric contents.

When Muhammad vomited in the prone position in response to the traumatic shock he was going into as a result of his asphyxial brain injury, his position and mechanical compression enabled and facilitated aspiration of his vomitus, which accentuated the mechanical-positional asphyxiation by initiating mechanical obstruction of his airways. The mechanical obstruction of his airways contributed and accentuated his spectrum of sustained mechanical-positional asphyxiation, and precipitated sudden and unexpected death.

The prevailing forensic scenario in this case confirms that Muhammad suffered two episodes of sustained compression of his trunk and body and mechanical obstruction of his airways within a short time. Muhammad suffered mechanical-positional asphyxiation for a cumulative period of approximately ten minutes [2 + 8 = 10].

The brain is about the only organ in the human body that exhibits the concept of cumulative injury, cumulative risk exposure to injury, or repetitive cumulative injury. Being a post-mitotic organ, if the brain suffers an injury, and within a relatively short time, suffers a repeat injury, the eventual outcome is an exponential and multiplicative combined effect of the two injuries, which increase the risk of sudden death. In traumatic brain injury, some refer to this concept as the second impact syndrome. This concept does not only apply to injuries suffered within a short time. For example, in football players who suffer Chronic Traumatic Encephalopathy [CTE] their risk of developing CTE is primarily based on their cumulative exposure to mild and seemingly innocuous sub-concussive and concussive blows to the head across time [years]. The risk of permanent and irreversible brain injury and death was exponentially increased when Muhammad suffered two episodes and two variants of asphyxiation within a relatively short time. Each episode and each variant possessed an independent and exclusive capacity and risk of causing permanent brain damage and death.

It is also pertinent to note that human beings who are suffering all forms of asphyxial injuries can perform phonation functions unless there is a direct early or preceding damage to the vocal cords and glottis. Some degrees of phonation may be expected, although impaired, but complete loss of phonation is not expected until there is paralysis of the glottis, directly or neurologically.

In the case of Muhammad, he suffered from Post-Traumatic Stress Disorder, Claustrophobia and Schizophrenia, minimal Coronary Artery Disease and Acute Amphetamine Intoxication in the morning of January 4, 2017. He was not in imminent death; he was not dying from any disease and was not expected to die. However, on January 4, 2017 a novel, independent and mutually exclusive, unnatural event occurred which comprised compression of his head, trunk, and extremities by multiple police officers while he laid prone on the hard ground for a cumulative period of 10 minutes. This high-scale traumatic stressor precipitated relaxation of his gastro-esophageal sphincter, which precipitated vomiting, which resulted in massive aspiration of gastric contents, which aggravated mechanical-positional asphyxiation by mechanical obstruction of the airways, which resulted in asphyxial brain injury and sudden death.

This novel chain of traumatic events successfully interrupted and breached any pre-existing chain of events and precipitated sudden death. The underlying cause of death therefore will be Mechanical-Positional Asphyxiation. The compression of his head, trunk, and extremities and his asphyxial brain injury were directly caused by another person or persons, therefore, the manner of death will be a homicide. There was a contiguous chain of events between the onset of compression of the trunk and body, and death.

In the differential diagnosis of the cause of death of Muhammad, there is no other forensic or medical evidence in this case that may suggest or indicate that Muhammad died from any other probable disease or cause of death outside Asphyxial Brain Injury due to Mechanical-Positional Asphyxiation.

If Muhammad did not encounter the police on January 4, 2017, he would not have died on January 4, 2017 and was not expected to die. The autopsy did not reveal any natural disease that was killing him. There is almost no adult who does not suffer from one form of ailment or the other. An absolutely normal human being is an anomaly. We all suffer from one ailment or the other including drug abuse, which is a disease. However, most of our ailments are treatable and manageable and do not expect to kill us at the young age of 43 years old.

2. Did Muhammad experience conscious pain and suffering before his death?

It is a generally accepted principle and common knowledge in medicine and forensic pathology, that specific traumatic events generate predictable, reproducible, and specific patterns of traumas and injuries. The patterns of injuries generated by blunt force trauma and asphyxial trauma, and the mechanisms of sustenance of these patterns of injuries are very well-established in the medical literature and are common knowledge.

Based on the prevailing forensic scenario, and on the generally accepted principles and common knowledge of medicine and science, and based on the global constellation, configurations and anatomic conformations of the multimodal and multifaceted traumas sustained by Muhammad, Muhammad experienced conscious pain and suffering when he sustained multiple blunt force traumas and asphyxial injuries on January 4, 2017 prior to his death.

Conscious pain and suffering are initiated by widespread free nerve endings situated in the skin, soft tissues, and organs. Pain can be elicited by multiple types of stimuli classified into three broad categories: mechanical, thermal, and chemical pain stimuli. Nerve endings for pain sensations generate electrical action potentials following all forms of tissue damage caused by all types of energies including, but not limited to, kinetic and mechanical energy from blunt force impacts, chemical and kinetic energy from mechanical compression of the body and obstruction of the airways from asphyxiation.

Action potentials are the sub-cellular physiologic basis for noxious conscious sensations and originate from voltage gated sodium and potassium electrolyte membrane pumps in the cell membranes of nerve cells, fibers, and synapses. It takes few 10,000th of a second to generate action potentials. Action potentials are transmitted through nerve fibers to the brain. They are transmitted in peripheral nerves in the Aδ and C fibers for fast and slow pain respectively at impulse rates of 5-30 meters per second and 0.5-2 meters per second, respectively. There is therefore a double pain sensation, a fast-sharp pain, and a slow pain. The sharp pain apprises the person rapidly of imminent danger and prompts the person to react immediately and remove himself from the painful stimulus or imminent danger. The slow pain becomes greater as time passes resulting in continued intolerable pain and suffering prompting the person to continue to try to relieve the cause of the pain and flee from the imminent danger.

At autopsy Muhammad measured 67 inches [1.70 meters]. Muhammad felt all types of blunt force, asphyxial and chemical pain within milliseconds of contact with an impacting surface, compression of the body, aspiration of food and asphyxiation. One millisecond is one second divided into 1000 parts. For the slowest nervous mechanisms of pain sensation and consciousness, a man like Muhammad felt pain in less than 100 milliseconds.

Nerve pathways transmitting pain, terminate in the spinal cord. Secondary pathways transmit the pain from the spinal cord to the brainstem and thalamus, especially to the reticular activating system of the brainstem. From the thalamus tertiary pathways transmit pain to other basal ganglia, limbic cortex, and neocortex of the brain. Pain stimuli are transmitted to the reticular nuclei of the midbrain, pons, and medulla; to the tectal midbrain and the periaqueductal gray matter. These lower regions of the brain, i.e. brainstem, are vital for the appreciation of suffering from pain.

Animals with their brains sectioned above the midbrain, to block any impulse reaching the neocortex and cerebral hemispheres, still experience suffering from pain caused by all types of trauma. Complete removal of the somatosensory regions of the cerebral hemispheres does not preclude an animal's ability to perceive and experience pain. Pain impulses entering the brainstem and lower centers of the human brain can cause conscious perception of pain. Pain perception is principally a function of the lower centers of the brain; however, the upper centers and cerebral hemispheres are responsible for the interpretation of the quality of pain.

Blunt force, compression of the body and asphyxiation elicit both the fast and slow pain types. Fast pain is felt within milliseconds while slow pain is felt within about one second. Following mechanical and chemical tissue damages and asphyxial tissue injury, biochemical tissue reactants like bradykinin, serotonin, histamine, prostaglandins, leukotrienes, potassium ions, substance P, acetylcholine, acids and proteolytic enzymes are expressed to elicit sustained secondary chemical pain in addition to the primary fast pain directly caused by tissue damages. The chemical pain elicited by these chemical reactants is a slow type of suffering pain. The intensity of pain is closely correlated with the rate of tissue damage.

The brain is responsible for and sustains consciousness in human beings. The region of the brain responsible for consciousness is the brainstem. The center in the brainstem, which is responsible for consciousness, is the reticular activating system, which is deeply located in the central regions of the brainstem. As long as the reticular activating system remains anatomically and electrochemically intact, an individual like Muhammad will remain conscious and will feel pain and experience suffering. The sensation of pain induces conscious suffering since pain is a noxious sensation, which stimulates the neocortex, limbic cortex, and forebrain to cause mental pain, suffering and anguish, and adrenergic fright and fear. All these neural processes occur in 1000th's of a second [milliseconds]. The human nervous system is one of the most efficient, effective, and optimal operating systems ever known to mankind. After centuries of empirical research mankind has not been able to fully decipher and reproduce the operating systems of the human brain and nervous system.

Muhammad's injury sustenance began when he was encountered by the police, held, pushed, tackled and forced to the ground, impacted by blunt force, held on the ground, with weight placed over him, vomited and aspirated his vomitus, which mechanically occluded his airways. At this time, Muhammad was fully conscious and aware of his surroundings. His reticular

activating center was completely intact and functional. As a 43-year-old male he had the mental capacity to identify the imminent danger and threat of injury and death.

The autopsy described at least 36 distinct blunt force injuries of Muhammad's head, face, trunk, and extremities. Every blunt force impact his body suffered, every kinetic and mechanical energy transferred to his body by the compression of his head, trunk and extremities, and every biochemical tissue response he endured from the asphyxial injury, caused biochemical, anatomic and pathophysiological tissue disruptions, damages and injuries generated action potentials within 10,000th of a second, which were transmitted to the spinal cord and to the brain to precipitate cumulative conscious somatic and mental pain, suffering and anguish. The multimodal nature of the noxious stimuli resulted in synergistic and cumulative conscious experience of very high levels of combined somatic and mental pain, suffering and anguish. The primary injuries initiated secondary tissue injuries, systemic and tissue reactive responses, which elicited novel chemical pain and accentuated the conscious somatic and mental pain, suffering and anguish.

The brainstem nuclei, the frontal cortex, pre-frontal cortex, basal forebrain and limbic cortex of Muhammad's brain initiated, within milliseconds, the primitive human reflexes of fear, fright, and flight. This mental awareness of imminent danger initiated the nor-adrenergic and adrenergic biochemical neural responses of fear, fright, and flight, when the locus ceruleus of the brainstem released large amounts of nor-adrenalin to the cerebral hemispheres. This fear, fright and flight response caused high levels of chemical mental pain, suffering and anguish, which prompted him to try to flee, struggle, yell, and scream, making noises of intense fright and mental anguish. His heart started pumping faster [chronotropic effect] and stronger [ionotrophic effect] due to the nor-adrenergic/adrenergic response. His respiratory rate and general muscle tonicity increased as well due to the nor-adrenergic/adrenergic response. His brain-cells' demands for oxygen increased causing intense cerebral vasodilation and vascular headache. All these patho-physiologic processes culminated in high levels of conscious somatic and mental pain, suffering and anguish.

As Muhammad was in this state of high-scale conscious somatic and mental pain, suffering and anguish, he received even more trauma when he vomited and aspirated his vomitus, which resulted in mechanical obstruction of his airways and aggravated the pre-existing mechanical asphyxiation and accompanying conscious somatic and mental pain and suffering. Additional millions of pain action potentials were generated and transmitted to the spinal cord and brain to cause even higher levels of conscious somatic and mental pain, suffering and anguish, which synergized with the existing somatic and mental pain, suffering and anguish, and even caused increasingly higher levels of conscious pain and suffering. Such high levels of conscious somatic and mental pain, suffering and anguish can impair the functioning of the limbic system and autonomic system of the brain and nervous system to cause an acute confusional and/or delirious state, which may have been exhibited by Muhammad.

As his blood oxygen levels decreased and the carbon dioxide levels increased, while he was on the ground, with his body compressed and pressed to the ground, he began to develop acidosis, became more confused, and afraid, and struggled more. His primary and secondary injuries progressed, more nerve endings in his body, soft tissues and viscerae were recruited, many more action potentials were elicited and caused increasingly higher levels of somatic pain and suffering, still accompanied by extremely high levels of mental pain, suffering and anguish. He gradually and slowly developed hypoxic-ischemic brain injury, which elicited more action

potentials from every brain tissue injury and damage, which resulted in more conscious somatic and mental pain, suffering and anguish as his sensorium diminished progressively.

As Muhammad's brain suffered hypoxic-ischemic injury many types of ions, peptides, proteins, and enzymes were expressed and activated, which enhanced his chemical pain, which synergized with his pre-existing pain to cause progressive somatic and mental pain, suffering and anguish. Following the attainment of irreversible brain damage, he began to lose consciousness progressively passing through the varying broad stages and spectrum of consciousness from fully conscious to varying degrees of diminishing sensorium to loss of consciousness and coma. Consciousness is a continuum with varying levels of consciousness and depths of unconsciousness, which Muhammad passed through before he went into traumatic coma and death.

The autopsy report confirms that Muhammad's brain and brainstem remained anatomically intact without any discernible physical brain damage. These evidentiary autopsy findings confirm that his midbrain, pons, and medulla oblongata remained anatomically intact following all his traumas until his death. The subcortical ganglia and brainstem nuclei of the cranial nerves remained intact. His rostral spinal and cranial reflexes and functioning remained anatomically intact. The reticular activating system remained anatomically intact. He continued to experience increasingly higher and extreme levels of somatic and mental pain, suffering and anguish induced by all his multimodal injuries until he attained irreversible brain injury and lost electrophysiological functioning of the brain cells, became unconscious and died.

It is pertinent to note that the autopsy did not describe any primary anatomic injury of the midbrain, pons, medulla oblongata, cerebral or cerebellar hemispheres. Injuries to these regions of the brain manifest as follows: primary lacerations, primary contusional hemorrhages and necrosis, primary gliding contusional hemorrhages and necrosis, primary concussional hemorrhages, primary diffuse traumatic axonal injury hemorrhages and necrosis. None of these distinctive evidence of primary anatomic trauma to the vital regions of the brainstem were present at autopsy. There was no evidence of instantaneous loss of consciousness at autopsy. There were no Adam's grade III concussional or contusional hemorrhages and necrosis, and no hemorrhages or necrosis of the dorsolateral and tegmental midbrain, pons, and medulla. Loss of consciousness was therefore sustained and progressed over time. After he lost consciousness Muhammad stopped experiencing conscious pain and suffering.

Loss of consciousness and death in this instance could not have been instantaneous since the human brain has an intrinsic ability to survive for a mean of about three to five minutes before suffering irreversible brain damage and brain death from any cause. The brain also has a metabolic reserve of about 5-10 seconds after suffering complete lack of oxygen and glucose before loss of consciousness ensues. Microscopic examination of the H/E stained sections of the brain and lungs confirm that Muhammad had enough time after he began suffering asphyxial injury for his tissues to develop patho-physiological responses to his injuries before he died.

Although death is frequently immediate, it is rarely instantaneous since death is a process that involves a cascade of patho-physiologic events. The adjective "immediate", within a forensic context, and within the prevailing forensic scenario, in this case, should be interpreted as death occurring as a result of mechanical-positional asphyxiation without the intervention of another object, cause, or factor. It should not be forensically construed as instantaneous.

Muhammad experienced conscious pain and suffering for a total composite median duration[13] of less than 15 minutes before he became unconscious on January 4, 2017 and died.

3. Did Methamphetamine prevent or preclude Muhammad from experiencing conscious pain and suffering?

No, Methamphetamine did not prevent or preclude Muhammad from experiencing conscious pain and suffering. The presence of Methamphetamine or Amphetamine at the time he sustained his fatal trauma, is of no significant forensic consequence in the differential diagnosis of his conscious pain and suffering.

Amphetamine is a metabolite of Methamphetamine. Methamphetamine or Amphetamine does not stimulate or inhibit naked nerve endings, which are the receptors that generate action potentials for pain sensation. Methamphetamine does not affect the conduction of action potentials in the central nervous system, to and from the spinal medulla and brain and does not modulate the experience of consciousness pain in any clinically useful manner. Methamphetamine or Amphetamine does not have analgesic properties and is not used as an analgesic drug to reduce or control pain.

The receptors for pain are the free nerve endings. So, the receptors for the mechanisms of generation and sensation of pain and suffering are different from the receptors and cellular sites for the mechanisms of action of Methamphetamine or Amphetamine. The generation and sensation of pain and suffering are complex mechanisms, which involve many different ions, peptides, and enzymes, including but not limited to bradykinin, histamine, potassium ions, acids, serotonin, acetylcholine, proteolytic enzymes, prostaglandins, and substance P.

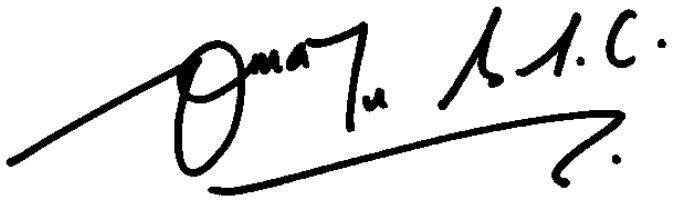
I have provided all my opinions and conclusions with a reasonable degree of medical certainty.

I reserve the right to amend, supplement, revise and/or modify my opinions and report, up and to the time of trial, should additional information become available.

Thank you.

Very truly yours,

[13] Medicine is not an absolute science and these estimated ranges should not be interpreted as absolute quantitative estimations of time. Quantitative ranges of any measurable index are common practice and are the standard of practice in pathology and medicine.



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